Effects of dry cow treatment of beef cows on pathogenic organisms, milk somatic cell counts, and calf growth during the subsequent lactation

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Effects of dry cow treatment of beef cows on pathogenic organisms, milk somatic cell counts, and calf growth during the subsequent lactation[1]

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ABSTRACT: Spring calving Angus and Angus × Hereford multiparous cows were utilized to determine the effects of intramammary treatment with penicillin G procaine (200,000 IU) and novobiocin (400 mg) at the time of weaning on udder health and calf growth after the subsequent calving. Cows were stratified by age and breed and assigned randomly to receive intramammary treatment (n = 99) at weaning or as untreated controls (n = 97). Quarter milk samples were collected at weaning and at 8 to 14 d after calving. Milk samples were analyzed for somatic cell counts (SCC) and mastitis-causing bacteria. Dry cow treatment decreased (P = 0.005) the number of cows infected after calving. Treatment decreased (P = 0.04) the number of cows that developed new infections and reduced (P = 0.03) the number of quarters with mastitis-causing bacteria after calving that were infected at weaning. Somatic cell counts after calving were greatest (P = 0.008) for cows infected with *Staphylococcus aureus*. Treatment did not alter (P = 0.19) SCC of quarters after calving that were infected with *S. aureus* at weaning but reduced (P = 0.002) SCC after calving of quarters that were infected with coagulase-negative staphylococci at weaning. Body weight of calves during early lactation was increased (P = 0.006) if cows with intramammary infection were treated at weaning. Treatment of noninfected cows at weaning increased (P = 0.008) adjusted 205-d weaning weights of calves after the subsequent lactation when compared with untreated noninfected cows. We conclude that treatment of beef cows at weaning with intramammary antibiotics decreased intramammary infections after calving, improved udder health during the subsequent lactation, and increased BW gain of the calves.

Key words: beef cattle, growth, mastitis, treatment

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INTRODUCTION

Weaning weight of calves is affected by age of dam, sex of calf, and forage intake (Neville, 1962; Cundiff et al., 1966; Ansotegui et al., 1991). Milk production of cows is the most important factor influencing BW gain of calves and accounts for about 60% of the variation in weaning weights (Neville, 1962; Rutledge et al., 1971). The influence of milk production on gain of calves is greatest during the first 60 d of age (Drewry et al., 1959; Neville, 1962), which corresponds to peak milk production in mature beef cows (Clutter and Nielsen, 1987; Marston et al., 1992).

Mastitis increases somatic cell counts (**SCC**) in milk and decreases milk production of dairy cows (Crossman et al., 1950; Bartlett et al., 1991; Lescourret and Coulon, 1994). Mastitis also causes increased SCC in beef cows (Watts et al., 1986; Simpson et al., 1995; Lents et al., 2002). Beef cows with greater SCC produced less milk (Simpson et al., 1995), and SCC are negatively correlated with weaning weights of calves (Watts et al., 1986). Thus, mastitis in beef cows is associated with decreased BW gain of calves (Haggard et al., 1983; Watts et al., 1986; Newman et al., 1991). Most intramammary infections occur during the dry period, and dry cow therapy is effective in eliminating udder infections during the dry period of dairy cows (Ziv et al., 1981; Davidson et al., 1994; Erskine et al., 1994). The objective of this experiment was to determine the effect of intramammary infusion of antibiotic in all quarters

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of beef cows at weaning on udder health, SCC, and calf growth during the subsequent lactation.

MATERIALS AND METHODS

Animals

All experimental procedures were approved by the Oklahoma State University Animal Care and Use Committee.

Spring calving, multiparous Angus, and Angus × Hereford cows (n = 196; mean age 5.3 ± 0.3 yr) were utilized to determine the effects of intramammary treatment with penicillin G procaine and novobiocin at weaning on udder infection and calf growth during the subsequent lactation. Different cows were treated during 2 yr (yr 1, n = 90; yr 2, n = 106). Cows grazed Bermudagrass and native grass pastures at the Oklahoma Agricultural Experiment Station Range Cow Research Center, 24 km west of Stillwater. At weaning in October, cows were stratified by age and breed and assigned randomly to intramammary treatment with antibiotic (n = 99) or untreated control (n = 97). Calves were weaned, and the cows were provided with a 40% CP supplement (1.4 kg each day on an as-fed basis) during the winter to maintain a moderate BCS (Wagner et al., 1988). Cows had a BCS of 4.9 ± 0.1 at calving. After the subsequent calving (February through May), calf BW were recorded at birth and every 30 d until weaning in October of yr 1. In yr 2, BW of calves was recorded at birth and at weaning in October.

Milk Samples and Treatment

Milk samples were collected from each quarter of each cow at weaning and again at 8 to 14 d after the subsequent calving. Calves were removed from the cows for approximately 2 h before sampling. Cows were administered 10 IU of oxytocin (i.m., Vedco Inc., St. Joseph, MO) to facilitate milk letdown. Teats were dipped in a 0.1% iodine solution (Alfa Laval, Agri Inc., Kansas City, MO) and dried with individual paper towels. The first 2 or 3 streams of milk were discarded, and 10 mL of milk from each quarter was collected into plastic vials containing preservative microtablets (D&F Control Systems Inc., San Ramon, CA). Samples were sent to the Dairy Herd Information Association laboratory (Manhattan, KS) within 24 h for analyses of SCC. Teat ends were then individually disinfected with a cotton swab soaked in 70% ethyl alcohol. Two streams of milk from each quarter were discarded, and 3 mL of milk was aseptically collected into sterile polypropylene snap-cap tubes (Fisherbrand, Pittsburgh, PA). Samples were placed on ice and transported to the laboratory and stored at −20°C until packaged in dry ice and transported to the Immunology and Disease Resistance Laboratory, USDA-ARS (Beltsville, MD) for bacteriological analyses.

Treatment was administered after both milk samples were collected at weaning. Ends of teats were individually wiped with alcohol pads, and 1 tube of penicillin G procaine and novobiocin (200,000 IU and 400 mg, respectively; Albadry Plus, Pharmacia & Upjohn, Kalamazoo, MI) was infused into each quarter. Albadry Plus is approved for treatment of mastitis in dry cows caused by *Staphylococcus aureus*, an organism consistently identified in our herd (Paape et al., 2000; Duenas et al., 2001; Lents et al., 2002). After treatment, teats were dipped in 0.1% iodine solution. Control cows were not infused, but their teats were dipped in iodine solution after collection of the milk samples.

Bacteriological Analyses

Bacteriological analyses were performed following standard procedures (NMC, 1990), with the exception that 20 μL of milk from each quarter of each cow was plated on 1 quarter of a 100 × 15-mm Petri dish containing esculin blood agar and on pyocyanin agar supplemented with acriflavine (Sigma Chemical Co., St. Louis, MO). Plates were incubated at 37°C, and bacterial growth was determined at 24 and 48 h. A quarter of the udder was considered to be infected if 3 or more colonies of the same organism were isolated from the esculin blood agar plate. Identification of organisms was based on colony morphology, hemolytic and hydrolytic patterns, Gram stain (Bacto Gram Stain Set, Difco Laboratories, Detroit, MI), catalase production (hydrogen peroxide, Sigma Chemical Co.), and tube coagulase test (coagulase plasma EDTA, Difco Laboratories). Tube coagulase tests were incubated at 37°C and examined for clot formation at 4 and 24 h. *Staphylococcus aureus* was identified by selective growth on pyocyanin agar supplemented with acriflavine and fermentation of mannitol. Coagulase-negative staphylococci (CNS) were further identified using an identification system for staphylococci (Api Staph, API-Bio Mérieux Vitex Inc., Hazelwood, MO). Bacteria were passed 4 times on esculin blood agar plates, and a second coagulase test was performed before Api Staph wells were inoculated. Streptococcal isolates were identified using cyclic adenosine monophosphate-esculin plates (Wilson et al., 1971) made in accordance with standard procedures (NMC, 1987). *Corynebacterium bovis* and *Bacillus* spp. were identified by time of appearance on incubated plates, colony morphology, and gram stain.

Statistical Analyses

Data were analyzed as a completely randomized design, and cow was the experimental unit when evaluating differences between cows, and quarter was the experimental unit when differences between quarters were evaluated. Cows were considered to be infected if 1 or more mastitis-causing organisms were present in 1 or more quarters. For a cow to be considered cured, mastitis-causing organisms could not be present in any quarter after calving. Effects of treatment and infection status at treatment on infection at calving were deter-

mined using PROC CATMOD (SAS Inst. Inc., Cary, NC). The model included year, treatment, infection status at treatment, and all 2- and 3-way interactions. Interactions were deleted from a reduced model if they were not significant ($P > 0.10$). Frequency tables for the number and percentage of cows and quarters infected for each level of each dependent variable were obtained using PROC FREQ of SAS. Bacteriological data for 17 infected and 18 noninfected cows after calving, from yr 2, were not available, because samples were lost during shipment. Milk samples from 1 additional cow in yr 2 were not available after calving. Missing data were equally distributed across all treatment groups, and there was no interaction of year with any main effect. Data from 160 cows that infection status was known both at treatment and calving were used to determine effects of treatment on infections after calving.

Somatic cell counts (thousands of cells/mL) were analyzed using log-transformed values (Ali and Shook, 1980; Lents et al., 2002); however, retransformed SCC are reported. Average SCC of the 4 quarters for each cow were determined as the geometric mean (Shook, 1982; Lents et al., 2002) and used as the average SCC value for each cow. The quarter with the greatest SCC for each cow was the maximum SCC value for that cow. Least squares ANOVA was used to determine treatment effects on SCC after calving. The model included year, treatment, infection status at weaning, and all 2- and 3-way interactions. If the interactions were not significant ($P > 0.10$), they were deleted from the reduced model.

In yr 1, calf BW were recorded each month from birth to weaning. Regression equations were developed using BW for each calf to determine the BW of the calf at 110 d. For analysis of weaning weights, data from yr 1 and 2 were used. Birth weight, weaning weight, and age were used to calculate an adjusted 205-d weaning weight. Effects of treatment on 110- and 205-d BW were determined using a model that included year (when appropriate), treatment, infection status at weaning, infection status at calving, sex, cow age, and significant ($P < 0.10$) 2- and 3-way interactions. Data for 87 calves were used in analysis of the 110-d BW, and data for 149 calves were used in analysis of the adjusted 205-d weaning weights.

RESULTS

Infection

Mastitis-causing bacteria isolated in this experiment were *S. aureus*, *C. bovis*, and CNS. The subspecies of CNS typically found in this herd have been reported (Paape et al., 2000; Lents et al., 2002). The percentage of cows and quarters that were infected at weaning were 28.8 and 10.9%, respectively. Of those, 20.6% of cows and 7.0% of quarters were infected with CNS, 8.1% of cows and 2.7% of quarters were infected with *S. aureus*, and 7.1% of cows and 2.7% of quarters were infected with *C. bovis*. Five cows were infected with both CNS and *S. aureus*, and 3 cows were infected with both CNS and *C. bovis* at weaning. Only 1 quarter of 1 cow was infected with more than 1 organism (CNS and *S. aureus*) at calving.

Table 1. Number of cows and quarters with infections at weaning (treatment) that cultured negative after calving[1] or that developed infections during the dry period

		Infected, n		Infections, n	
Unit	n	Weaning	Calving	NC[2]	Developed
Cows					
Control	81	24	27[a]	12	15[a]
Treated	79	22	13[b]	16	7[b]
Quarters					
Control	316	35	43[a]	24[a]	32[a]
Treated	310	33	17[b]	32[b]	16[b]

[a,b]Means within a unit and column with different superscript letters differ ($P < 0.05$).
[1]Based on bacterial growth from a single aseptically collected milk sample.
[2]NC = negative culture after calving that was positive at treatment.

The number of cows infected at treatment (weaning) was not different ($P = 0.51$) for treated and control cows (Table 1). The number of cows infected at calving was less ($P < 0.001$) in yr 2 than in yr 1 (8 vs. 32, respectively). There was no interaction of year with treatment for infection at calving ($P = 0.31$), so data were pooled across years to test the effect of treatment and infection status at weaning on the presence of mastitis-causing bacteria at calving. Infection status at weaning did not influence ($P = 0.19$) the response to treatment. Treatment at weaning reduced ($P = 0.005$) the number of cows infected at the subsequent calving (Table 1). The number of cows with infections that were cured during the dry period (based on bacterial growth from a single aseptically collected milk sample) was not affected ($P = 0.18$) by treatment; however, treatment reduced ($P = 0.04$) the number of cows that developed new infections (Table 1). When data were analyzed based on individual quarters, treatment increased ($P = 0.002$) the number of infected quarters that were cured during the dry period (Table 1). Treatment also reduced ($P = 0.01$) the number of quarters that developed new infections during the dry period (Table 1).

The effect of treatment on infection postpartum differed with the type of mastitis-causing organism present. Treatment at weaning reduced the number of cows ($P = 0.01$) and quarters ($P = 0.02$) infected with CNS at calving but did not affect the number of cows ($P = 0.16$) or quarters ($P = 0.55$) infected with *S. aureus* (Table 2). Infection status of a cow or quarter at weaning influenced ($P = 0.048$) the incidence of infection at the subsequent calving (Table 3). A greater number of cows ($P = 0.01$) and quarters ($P = 0.02$) that were infected at weaning were infected at calving compared with the number that were not infected at weaning and were infected at the next calving (Table 3). This was associ-

Table 2. Number of cows and quarters with coagulase-negative staphylococci (CNS) or *Staphylococcus aureus* infections at weaning and after calving

Unit	n	Number infected with CNS		Number infected with *S. aureus*[1]	
		Weaning	Calving	Weaning	Calving
Cows					
Control	81	17	21[a]	8	9
Treated	79	16	9[b]	5	4
Quarters					
Control	316	21	27[a]	11	12
Treated	310	23	12[b]	6	5

[a,b]Means within a unit and column with different superscript letters differ ($P < 0.05$).
[1]Two quarters that were infected with *S. aureus* at treatment were not functional at calving.

ated with a greater ($P < 0.001$) percentage of cows and quarters infected with *S. aureus* at treatment that remained infected at calving. Infection with CNS at weaning did not influence ($P = 0.43$ and 0.89 for cows and quarters, respectively) the occurrence of CNS infections at the subsequent calving.

There was a year effect ($P < 0.001$) on the incidence of *C. bovis* at weaning. There were no *C. bovis* infections at weaning in yr 1. In yr 2, *C. bovis* was found in 14 cows and 21 quarters at treatment. However, data for 8 cows and 12 quarters were not available at the subsequent calving due to loss of samples during shipment. This precluded statistical analysis of treatment effects on *C. bovis* infections, but observations are summarized for cows and quarters for which bacteriological analyses were available at calving. At weaning, 3 control cows and 3 control quarters were infected with *C. bovis*.

Table 3. Influence of infection status at weaning on the percentage of cows and quarters infected at calving

Bacteria type[1] at weaning	Infection status at weaning	Infected at calving, %
Any bacteria		
Cows	Not infected	20.2[a]
	Infected	39.1[b]
Quarters	Not infected	8.7[a]
	Infected	17.7[b]
CNS		
Cows	Not infected	18.1
	Infected[1]	24.2
Quarters	Not infected	6.2
	Infected	6.8
S. aureus		
Cows	Not infected	4.1[a]
	Infected	53.9[b]
Quarters	Not infected	1.9[a]
	Infected	33.3[b]

[a,b]Column means within bacteria type and unit with different superscript letters differ ($P < 0.05$).
[1]Any bacteria includes coagulase-negative staphylococci (CNS), *Staphylococcus aureus*, and *Corynebacterium bovis*. Three cows infected with CNS were also infected with *C. bovis*.

Table 4. Effect of type of mastitis-causing bacteria present at sampling on average somatic cell counts (SCC) per cow

Time and type of bacteria	Cows, n	Average SCC,[1] thousands of cells/mL	SEM
Weaning			
None	129	34.5[a]	12.3
C. bovis	11	20.3[a]	42.2
CNS[2]	35	145.6[b]	23.7
S. aureus[3]	13	192.2[c]	38.8
Calving			
None	113	61.7[a]	19.5
CNS[2]	25	104.3[a]	40.6
S. aureus[3]	13	308.2[b]	57.4

[a–c]Means within a sampling time with different superscript letters differ ($P < 0.05$).
[1]Average SCC = geometric mean for SCC of all quarters of a cow.
[2]CNS = coagulase-negative staphylococci. Includes cows infected with both CNS and *Corynebacterium bovis*.
[3]Includes cows infected with both *Staphylococcus aureus* and CNS.

These infections were cured during the dry period while 3 additional control cows and 5 additional control quarters developed new *C. bovis* infections. Three treated cows and 6 treated quarters were infected with *C. bovis* at weaning, and all of these infections were cured during the dry period. No new *C. bovis* infections occurred in treated cows or quarters. When *C. bovis*-infected cows were combined with CNS-infected cows and reanalyzed as cows infected with total minor mastitis-causing pathogens, results were similar (data not shown) to when CNS-infected cows were analyzed separately.

SCC

Average SCC of cows at weaning and at the subsequent calving were increased ($P = 0.046$) by the presence of mastitis-causing bacteria. Average SCC of cows infected with both CNS and *S. aureus* were greater ($P = 0.03$) than for cows infected with only CNS, and SCC for cows infected with only *S. aureus* were similar ($P = 0.32$) to numbers for cows infected with both CNS and *S. aureus*. Thus, average SCC of cows infected with both *S. aureus* and CNS were pooled with those for cows infected with only *S. aureus* for analysis. Similarly, average SCC of cows infected with both CNS and *C. bovis* were similar ($P = 0.49$) to those of cows infected with only CNS but greater ($P = 0.08$) than for cows infected with *C. bovis* alone. Average SCC for cows infected with both *C. bovis* and CNS were pooled with those of cows infected with CNS alone. At weaning, average SCC were greatest ($P = 0.02$) for cows infected with *S. aureus* (Table 4). Cows infected with CNS at weaning had greater ($P < 0.001$) average SCC at treatment than cows infected with *C. bovis* or noninfected but had less ($P = 0.02$) average SCC than cows infected with *S. aureus* (Table 4). Average SCC at treatment were not different ($P = 0.17$) for cows infected with *C. bovis* compared with noninfected cows (Table 4). At the subsequent calving, average SCC were not ($P = 0.89$)

Table 5. Effect of type of mastitis-causing bacteria present at sampling on somatic cell counts (SCC) per quarter

Time and type of bacteria	Quarters, n	Quarter SCC, thousands of cells/mL	SEM
Weaning			
None	640	56.7[a]	19.7
Corynebacterium bovis	21	50.1[a]	110.0
CNS[1]	55	775.2[b]	67.1
Staphylococcus aureus	17	1,142.6[c]	120.6
Calving			
None	534	139.5[a]	25.0
CNS	38	332.6[b]	94.9
S. aureus	14	1,244.1[c]	154.4

[a–c]Means within a sampling time with different superscript letters differ ($P < 0.05$).
[1]CNS = coagulase-negative staphylococci.

Table 6. Effect of treatment and infection status at weaning on somatic cell counts (SCC; thousands of cells/mL) of cows and quarters at the subsequent calving

Unit	Control	Treated	SEM
Cow			
Average SCC[1]	105.4	114.8	25.9
Maximum SCC[2]	636.2	384.5	122.7
Quarter SCC[3]			
Not infected at weaning	186.1[a]	154.4	37.5
Infected at weaning	438.7[b]	91.2	106.4

[a,b]Means within a unit and column with different superscript letters differ ($P < 0.05$).
[1]Average SCC = geometric mean for SCC of all quarters of a cow.
[2]Maximum SCC = the quarter of a cow with the maximum somatic cells.
[3]Treatment × infection status at drying-off ($P < 0.05$).

different for cows infected with CNS and noninfected cows (Table 4). Average SCC at calving were greatest ($P < 0.001$) for cows infected with *S. aureus* (Table 4).

Quarters infected with mastitis-causing bacteria had greater ($P < 0.001$) SCC than noninfected quarters at weaning (770 ± 53 and 58 ± 20 × 10^3 cells/mL, respectively) and at calving (681 ± 86 and 149 ± 25 × 10^3 cells/mL, respectively). The type of mastitis-causing bacteria present at sampling affected ($P = 0.038$) SCC per quarter. Somatic cell counts per quarter at weaning were not different ($P > 0.31$) for noninfected quarters and quarters infected with *C. bovis* (Table 5). Quarters infected with CNS had greater ($P = 0.008$) SCC than noninfected quarters or quarters infected with *C. bovis* (Table 5). Quarters infected with *S. aureus* at weaning had the greatest ($P = 0.042$) SCC (Table 5). At calving, SCC were greater for quarters infected with CNS compared with noninfected quarters ($P = 0.032$), and SCC were greatest ($P = 0.007$) for quarters infected with *S. aureus* (Table 5).

There were no interactions between year or infection status at weaning with treatment for average SCC or maximum SCC at weaning. Thus, data were pooled across years and infection status at weaning, and the main effect of treatment on average SCC and maximum SCC are presented. There were no differences in average SCC ($P = 0.41$) and maximum SCC ($P = 0.27$) at weaning for treated and control cows.

Average SCC at calving were greater ($P = 0.014$) in yr 2 than yr 1 (115 ± 24 vs. 105 ± 28 × 10^3 cells/mL, respectively), but there were no interactions of year ($P = 0.27$) or infection status at weaning ($P = 0.41$) with treatment for average SCC or maximum SCC of cows after calving. Treatment at weaning did not reduce average SCC ($P = 0.16$) or maximum SCC ($P = 0.14$) per cow at the subsequent calving (Table 6).

Treatment did not influence ($P = 0.17$) SCC for quarters at weaning (388 ± 33 and 439 ± 34 × 10^3 cells/mL for treated and control cows, respectively). Somatic cell counts of quarters at calving were greater ($P < 0.001$) in yr 2 than yr 1 (233 ± 48 and 202 ± 46 × 10^3 cells/mL, respectively). However, there was no interaction of year with treatment ($P = 0.32$) or infection status at weaning ($P = 0.21$) on SCC of quarters at the subsequent calving. There was an infection status at weaning × treatment effect ($P = 0.002$) on SCC of quarters at the subsequent calving. Somatic cell counts were greater ($P < 0.001$) in control quarters that were infected at weaning compared with control quarters that were not infected at weaning (Table 6). Treatment did not influence ($P = 0.70$) SCC of quarters at calving regardless of infection status at treatment (Table 6). Treatment decreased ($P = 0.002$) SCC at calving of quarters that were infected with CNS at weaning (317 ± 136 vs. 97 ± 126 × 10^3 cells/mL for control and treated CNS-infected quarters, respectively). Treatment did not alter ($P = 0.19$) SCC of quarters at calving that were infected with *S. aureus* at weaning.

Calf BW

There were no interactions between intramammary infection status of the cow at weaning and treatment ($P = 0.51$), and infection status of the cow at weaning and infection status at calving ($P = 0.32$), on BW of calves at 110 d of age. There was an interaction ($P = 0.03$) between treatment and infection status of the cow at calving on BW of calves at 110 d. If cows were not infected at calving, BW of calves at 110 d were similar ($P = 0.97$) for control and treated cows. However, if cows were infected at calving, BW of calves at 110 d were greater ($P = 0.006$) for treated than control cows (Table 7).

Adjusted 205-d weaning weights were greater ($P < 0.05$) in yr 1 than yr 2 (243.3 ± 2.7 vs. 207.8 ± 3.2 kg, respectively), but there was no interaction of year with treatment ($P = 0.28$). There were no interactions between treatment at weaning and infection status of cows at calving ($P = 0.48$) and between infections status at weaning and infection status at calving ($P = 0.52$). There was an interaction between treatment and infection status of cows at weaning ($P = 0.03$) on adjusted 205-d weights. Adjusted 205-d weights at the end of

Table 7. Least squares means for weight of calves at 110 d during the subsequent lactation for control and treated cows that were infected or not infected at calving

Treatment	Calf weight at 110 d[1] kg	SE
Control		
Not infected	144.8[ab]	4.0
Infected	137.4[a]	3.8
Treated		
Not infected	144.4[ab]	3.4
Infected	157.4[b]	6.4

[a,b]Means (for both treatments) with different superscript letters differ ($P < 0.05$).
[1]Treatment × infection at calving ($P = 0.03$).

the subsequent lactation were similar ($P = 0.77$) for control cows regardless of their infection status at the previous weaning (Table 8). However, treated cows that were not infected with mastitis-causing organisms at weaning had calves with greater ($P = 0.008$) adjusted 205-d weights after the subsequent lactation than treated cows that were infected at weaning (Table 8).

DISCUSSION

Rate of new intramammary infections is greater in dairy cows during the dry period as compared with during lactation (Neave et al., 1950; Oliver and Mitchell, 1983). The purpose of dry cow therapy is to eliminate existing infections and prevent new infections from occurring. Treatment of dairy cows at drying off with antibiotics decreases infections at the next lactation (Harmon et al., 1986, Hogan et al., 1994) and prevents new infections during the dry period (Cummins and McCaskey, 1987; Batra, 1988; Hogan et al., 1994). In the current study, dry cow treatment decreased the number of infected cows and quarters at the subsequent calving. This was primarily due to a decrease in the occurrence of new infections during the dry period. Newman et al. (1991) found that antibiotic treatment of beef cows did not influence prevention of new infections during the dry period. The difference in the ability of dry cow treatment to prevent infections at calving could be related to the treatment protocol. Newman et al. (1991) only treated cows with quarters that were determined to be infected. In our experiment, cows were assigned randomly to treatment without knowledge of infections status or SCC of a quarter. With this approach, noninfected quarters received treatment and were protected against development of infections during the dry period. In dairy cows, most new infections occur within weeks after drying-off or immediately before lactation (Neave et al., 1950; Oliver and Mitchell, 1983). Additionally, dry cow treatment of dairy cows prevented new intramammary infections at 4 to 10 d after calving (Sinkevich et al., 1974). In our experiment, beef cows were sampled 8 to 14 d after calving, a time when the protective benefits of dry cow treatment might be evident. In addition, the antibiotic used for treatment could influence results. A penicillin-novobiocin product was used in the current experiment, and Newman et al. (1991) used cephapirin benzathine. Seymour et al. (1989) found that cephapirin did not influence infection status of dairy cows during the dry period, and cephalonium, a cephapirin analog, was less effective than other antibiotics in preventing the development of new infections in dairy cows (Ziv et al., 1981).

Table 8. Least squares means for adjusted 205-d weaning weight of calves for control and treated cows that were infected or not infected at the previous weaning

Treatment	Adjusted 205-d weaning weight kg	SE
Control		
Not infected	222.2[a]	3.1
Infected	223.8[a]	4.4
Treated		
Not infected	235.7[b]	3.5
Infected	220.5[a]	4.9

[a,b]Means (for both treatments) with different superscript letters differ ($P < 0.05$).

Dry cow treatment of dairy cows increases cure rates of existing infections (Ziv et al., 1981; Harmon et al., 1986; Schukken et al., 1993). Treatment did not increase cure rate of infections present at weaning in the current experiment when evaluated on the basis of individual cows. When cure rate was evaluated on the basis of individual quarters, treatment was effective in eliminating infections present at weaning. This is consistent with previous results for infected quarters of beef cows (Newman et al., 1991). By definition, for a cow to be considered cured, mastitis-causing organisms could not be present in any quarter after calving. Some cows had an infected quarter that was cured during the dry period but became infected in another quarter and thus were not classified as cured. Two cows were infected in a single quarter with *S. aureus* at treatment, and the infection persisted during the dry period. Each of these quarters was not functional at calving, and these cows were not classified as cured. Clinical signs of mastitis were not determined in this study, and some cows with pathogenic organisms probably did not have clinical mastitis. Cure rates were similar for treated and control dairy cows that had subclinical symptoms of mastitis (Seymour et al., 1989).

The incidence of infection is consistent with our previous reports for this herd (Paape et al., 2000; Lents et al., 2002). Dry cow treatment reduced the number of cows and quarters infected with mastitis-causing organisms after calving. This is consistent with previous reports for dry cow treatment of beef cows (Newman et al., 1991). The beneficial results of treatment were due primarily to elimination of minor mastitis-causing

pathogens such as CNS. In contrast, *S. aureus* infections persisted through the dry period, and dry cow treatment was not effective in altering the number of quarters infected with *S. aureus* at calving. Infection with *S. aureus* may result in fibrotic tissue encapsulating the infection, and antibiotics cannot reach the site, making it difficult to cure (Nickerson and Owens, 1993). Administering systemic antibiotic to beef cows at drying-off, and again at calving, reduced the incidence of new infections of *S. aureus* (Duenas et al., 2001).

Somatic cell counts were greatest for quarters infected with *S. aureus* and intermediate for quarters infected with CNS. Infected quarters of beef cows average approximately 500 to 800 $\times$ 10^3 somatic cells/mL (Hunter and Jeffrey, 1975; Paape et al., 2000; Lents et al., 2002). Somatic cell count was not increased in *C. bovis*-infected quarters at weaning. Somatic cell counts in CNS-infected quarters were only modestly increased; thus, average SCC at calving for cows infected with CNS were not different from average SCC of noninfected cows. The minimal increase in SCC of CNS-infected quarters may have a role in preventing development of infections caused by major pathogens (Linde et al., 1975; Nickerson and Boddie, 1994) by providing a greater initial immune response to infection (Rainard and Poutrel, 1988; Matthews et al., 1990, 1991). Wilson et al. (1971) observed that although mastitis-causing bacteria may be present in beef cows, SCC may not be elevated to abnormal concentrations. The lack of increased average SCC of cows infected with CNS or *C. bovis* indicate that beef cows infected with minor mastitis-causing pathogens may not show clinical symptoms. In contrast, quarters infected with *S. aureus* had greater SCC than quarters infected with minor pathogens. Thus, cows infected in at least 1 quarter with *S. aureus* had increased average SCC per cow. This further substantiates our previous report that *S. aureus* infection causes greater quarter SCC for beef cows than do minor pathogens (Paape et al., 2000).

Although others have also evaluated the effect of dry cow treatment of beef cows on udder infections (Newman et al., 1991), they did not report the effect of treatment on SCC. Herein administering antibiotics at weaning did not decrease average SCC of cows at calving compared with controls. Treatment also failed to reduce the maximum SCC of cows after calving. This is most likely due to the fact that most quarters of cows with increased SCC were infected with *S. aureus*, which was not influenced by treatment. When evaluated on the basis of all individual quarters, there was an interaction between treatment and infection status at weaning. If quarters were not infected at weaning, SCC at calving were similar for treated and control quarters. However, if quarters were infected at weaning, treatment reduced SCC at calving. This beneficial effect could be attributed to the reduction of CNS infections of treated quarters during the dry period.

Although mastitis has been studied in beef cows, few researchers have attempted to evaluate effects of intramammary treatment of cows on BW gain of calves. The negative correlation between milk production and udder infection that occurs in dairy cows is expected to exist in beef cows (Simpson et al., 1995). Beef cows with udder infections had calves with decreased BW gains (Haggard et al., 1983; Newman et al., 1991; Simpson et al., 1995), and intramammary dry cow treatment decreased udder infections during the subsequent lactation (Newman et al., 1991). However, these authors did not evaluate effects of dry cow therapy of cows on performance of calves during the subsequent lactation. Calves from cows treated for mastitis at drying-off weighed 12.5% more at 60 d than calves from untreated controls (Kirkbride, 1977). We previously determined that treatment of cows with i.m. antibiotics at drying-off and again after calving decreased udder infections (Duenas et al., 2001) but did not alter BW (Lents et al., 2002). In the current study, there was a significant interaction between treatment and infection status of the cow at calving on BW of calves during the next lactation. Weight of calves at 110 d was similar for treated and control cows if they were not infected at calving. However, if cows were infected at calving, BW of calves at 110 d was greater for treated than control cows. Administering antibiotics to cows at drying-off may provide a prophylactic effect at the beginning of the subsequent lactation in beef cows. This treatment may have improved udder health and supported greater BW gain of calves early in lactation. Adjusted 205-d weaning weight of calves was also influenced by antibiotic treatment before the previous dry period. Calves from cows that were treated but not infected at weaning had greater adjusted 205-d weaning weights during the subsequent lactation. Taken together with the observations that dry cow treatment decreased the incidence of udder infection and increased BW gain of calves, this supports the hypothesis that improved udder health during the next lactation after dry cow therapy could support greater BW of calves at weaning.

Dry cow therapy decreases udder infections of beef cows at the subsequent calving by eliminating infections that exist at drying-off and reducing the incidence of new infections during the dry period. The SCC of quarters infected with mastitis-causing bacteria at drying-off was reduced with dry cow therapy, indicating that dry cow treatment improves udder health of the beef cow. Administering intramammary antibiotics to beef cows at drying-off can reduce the incidence of udder infections in beef cows after calving and increase BW gain of calves during the subsequent lactation.

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